



# Subject : Pathology

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## HEMODYNAMIC DISORDERS 2023-2024 AUDIO 1-2



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#### **OBJECTIVES OF HEMODYNAMICS**

- Hyperemia
- Congestion and Edema
- Types and causes of edema
- Hemorrhage (types of hemorrhage)
- Thrombosis & DIC
- Embolism. (Types of Embolism )
- Infarction.(Types of Infarction)
- White infarction
- Red infarction
- Shock(Types of shock)
- Hypovolemic shock, Septic Shock
- Anaphylactic shock
- Neurogenic shock
- Cardiogenic shock

#### INTRODUCTION

- Hemodynamic disorders are very common & extremely important cause of clinical illnesses.
- The health of cells & tissues depends on the circulation of blood , which delivers oxygen & nutrients and removes wastes generated by cellular metabolism .
- Under normal conditions, as blood passes through capillary beds, proteins in the plasma are retained within the vasculature and there is little movement of water & electrolytes into the tissues.
- This balance is often disturbed by pathologic conditions that Calter endothelial cells function, increase vascular pressure, or
   decrease plasma protein content, all of which promote edema i.e. accumulation of fluid in extra vascular spaces.

- Hemostasis is the process of blood clotting that prevents excessive bleeding after blood vessel damage.
  - Hemostasis is the mechanism that leads to cessation of bleeding from a blood vessel. It is a process that involves multiple interlinked steps. This cascade culminates into the formation of a "plug" that closes up the damaged site of the blood vessel controlling the bleeding
- Inadequate hemostasis may result in hemorrhage which can affect tissue perfusion & if its massive and rapid; it may lead to hypotension, shock & death.
- Conversely, inappropriate clotting i.e. thrombosis or migration of clot called embolism can obstruct blood vessels causing ischemic cell death i.e. Infarction.
- Thrombo-embolism lies at the heart of three major causes of morbidity & death in developed countries, myocardial infarction, pulmonary embolism & cerebro-vascular accidents (CVA) or stroke.

#### قراءة للفهم ؟ .

Hemostasis is the intricate process that prevents excessive bleeding after blood vessel damage. It involves a cascade of interlinked steps culminating in the formation of a clot, which acts as a plug to stop bleeding. Inadequate hemostasis can lead to hemorrhage, affecting tissue perfusion and, if severe, may result in hypotension, shock, and death.

Conversely, inappropriate clotting, such as thrombosis or embolism (migration of a clot), can obstruct blood vessels, causing ischemic cell death known as infarction. Thromboembolism is central to major causes of morbidity and death in developed countries, including myocardial infarction, pulmonary embolism, and cerebrovascular accidents (CVA) or stroke. It highlights the delicate balance required for proper blood clotting to maintain health and prevent life-threatening complications.

## HYPEREMIA & CONGESTION

 Both terms, hyperemia & congestion, indicate increased local blood volume in a particular tissue,

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- But Hyperemia is an active process, resulting from increased blood flow due to arteriolar dilation, at sites of inflammation or in skeletal muscle during exercise, & the hyperemic tissue is red.
- Congestion is a passive process, resulting from impaired venous return from a tissue. The congested tissue is cyanotic, bluish-red in color because congestion leads to accumulation of deoxygenated hemoglobin in the congested tissues





Figure : Photographic appearance of hyperemia of the inflammed conjunctiva of eye .



Figure : Gross view of hyperemia of the brain , brain looks reddish .

### **Congestion** may be

- systemic, as in Congestive heart failure,
   or localised resulting from an isolated venous obstruction.
- Congestion & edema commonly occur together.
- In long- standing chronic venous congestion (CVC), the stasis of poorly oxygenated blood ( causes chronic hypoxia , which can result in paranchymal cell degeneration or death, & subsequent tissue fibrosis.
  - Capillary rupture at sites of CVC may also cause small foci of hemorrhage.

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#### **Pulmonary congestion :**

 Grossly: The congested lung is heavy, and dark red in lood stained fluid will be squeezed out.
 Microscopically :

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- Acute pulmonary congestion is characterized by alveolar capillary distension with blood, alveolar septal edema, and/or focal minute intra-alveolar hemorrhage.
- While in chronic pulmonary congestion, the alveolar septa become thickened & fibrotic & the alveolar spaces may contain numerous hemosiderin–laden macrophages, so-called *("heart failure cells")*.



In chronic pulmonary congestion, prolonged exposure to increased blood pressure in the pulmonary circulation leads to changes in the lung's structure. The alveolar septa, which are the thin walls that separate the air sacs (alveoli) in the lungs, undergo thickening and fibrosis. This fibrotic process is a result of the chronic stress placed on the lung tissues due to the congestion.

Within the alveolar spaces, there is an accumulation of hemosiderin-laden macrophages. Hemosiderin is a pigment derived from the breakdown of red blood cells. The presence of these macrophages gives rise to the term "heart failure cells." These cells are indicative of a condition where blood leaks into the alveoli due to increased pressure in the pulmonary blood vessels.

The hemosiderin-laden macrophages are a sign of repeated episodes of bleeding within the lungs. As blood leaks into the alveoli, macrophages engulf the hemosiderin from the breakdown of red blood cells. The term "heart failure cells" reflects the association of these changes with chronic heart failure, as congestive heart failure is often accompanied by pulmonary congestion and the described alterations in lung tissue.



Figure - Gross view of acute lung congestion, lung tissue is dark red with frothy fluid comes out during cutting.



#### Figure : Normal lung histology



Figure: Microscopic view of acute pulmonary congestion, showing congested capillaries in alveolar septa with intra-alveolar edema (arrow).



Figure: Chronic pulmonary congestion showing golden-yellow appearance of hemosiderine –laden macrophages i.e. (Heart-failure cells) & fibrosis of alveolar septa.

### **LIVER CONGESTION**

- Congestive hepatopathy is diffuse venous congestion within the liver that results from right-sided heart failure (usually due to cardiomyopathy, tricuspid regurgitation, mitral insufficiency, cor pulmonale, or constrictive pericarditis).
  Grossly: Microscopically
- There is centri lobular hepatic cell necrosis & hemorrhage, with hemosiderinladen macrophages, alternating with pale peripheral zones of fatty change in peripheral hepatocytes. In severe & and long-standing hepatic Central Venous Congestion(commonly due to heart failure), there may even be grossly evident hepatic fibrosis, so-called "cardiac cirrhosis". cirrhosis due to the congestive Heart Failure In chronic venous congestion the liver has a nutmeg-like appearance, because the central portions of the hepatic lobule are the last to receive blood from both the portal vein & hepatic artery, so they tend to undergo early necrosis due to ischemic injury, whenever there is reduced hepatic blood flow with hemorrhage thus look dark red & peripheral zone look pale, due to unaffected hepatocytes or fatty changes.

Congestive hepatopathy is characterized by diffuse venous congestion in the liver, typically resulting from right-sided heart failure caused by conditions like cardiomyopathy, tricuspid regurgitation, mitral insufficiency, cor pulmonale, or constrictive pericarditis.

Grossly, the liver exhibits centrilobular hepatic cell necrosis and hemorrhage. Hemosiderin-laden macrophages, indicative of previous bleeding, alternate with pale peripheral zones showing fatty changes in hepatocytes. In severe and prolonged cases, there may be evident hepatic fibrosis, termed "cardiac cirrhosis."

In chronic venous congestion, the liver displays a nutmeg-like appearance. This is due to the central portions of the hepatic lobule being the last to receive blood from both the portal vein and hepatic artery. These central areas are prone to early necrosis from ischemic injury when there is reduced hepatic blood flow. Consequently, these central regions appear dark red due to hemorrhage and necrosis, while the peripheral zones look pale, either due to unaffected hepatocytes or fatty changes. This nutmeg appearance reflects the complex interplay of altered blood flow and tissue damage in the liver associated with chronic venous congestion.

مرض الكبد الاحتقاني يتميز بتكون احتقان واسع في الأوعية الوريدية داخل الكبد، وغالباً يكون نتيجة لفشل القلب الجانبي الأيمن والذي ينجم عن حالات مثل توسع العضلة القلبية (القلب العضلي)، تسرب الدم من صمام الرئة الثلاثي (تسرب الدم عبر الصمام التاجي)، عدم كفاية الصمام الميترال، توسع القلب الرئوي، أو التهاب الأعضاء الغشائية المحيطة بالقلب (التهاب الحقيبة القلبية).

من الناحية الظاهرية، يظهر الكبد بانتكاس حاد في خلاياه المركزية مع نزيف. تحمل خلايا الماكروفاج المشبعة بصبغة الهيموسيدرين، التي تشير إلى نزيف سابق، يتناوب مع مناطق هلامية في الحواف الطرفية تظهر تغيرات دهنية في خلايا الكبد. في حالات شديدة ومستمرة، قد يظهر تليف واضح في الكبد، ويُصطلح على هذا بـ "تليف القلب".

في الاحتقان الوريدي المزمن، يظهر الكبد مظهراً شبيهاً بالجوز نتيجة للأجزاء المركزية للفص الكبدي تكون آخر من تتلقى الدم من الوريد الكبدي والشريان الكبدي. هذه المناطق المركزية عرضة للتلف المبكر بسبب الإصابة الناتجة عن النقص في تدفق الدم الكبدي. وبناءً على ذلك، تظهر هذه المناطق المركزية بلون أحمر غامق بسبب النزيف والتلف، بينما تظهر المناطق الطرفية بلون باهت، إما بسبب خلايا الكبد غير المتأثرة أو التغيرات الدهنية. هذا المظهر العنو يعكس التفاعل المعقد بين تغيرات تدفق الدم والضرر النسيجي في الكبد المرتفي الاحتقان الرم الكبدي. وبناءً على ذلك



Figure : Normal liver , microscopic view .



Figure : Nut meg liver in chronic venous congestion due to heart failure .A-Nut meg grain . Increased systemic venous pressure from any cause lead to hepatic

cingestion, liver becomes enlarged, tender & pulsatile



Figure: Nut meg liver in CVC, gross view. The gross pathological appearance of a liver affected by chronic passive congestion is "speckled" like a grated nutmeg; the dark spots represent the dilated and congested hepatic venules and small hepatic veins. The paler areas are unaffected surrounding liver tissue



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# Figure: Gross & microscopic appearances of liver in CVC.

Figure: microscopic view of the liver in CVC (nutmeg liver )showing necrotic hepatocytes & hemorrhage around the central vein giving red color to this area.



- 60% of the lean (without fat) body weight is water, with 2/3 intracellular (within cells); & 1/3 extracellular (outside the cells), mostly as interstitial fluid.
- 5% of total body water only is in the intravascular compartment, i.e. in the blood plasma.
- The term edema refers to increased fluid in the interstitial tissue spaces.
  - Fluid collections in different body cavities are referred to as :

**Hydrothorax** : in pleural cavity. **Hydropericardium**: in the pericardial cavity.

Hydroperitoneum: in the peritoneal cavity also called ascites.

Anasarca: Is a severe generalized edema with a profound subcutaneous swelling .

The human body is composed of approximately 60% water in terms of lean (non-fat) body weight. Two-thirds of this water is located intracellularly (within cells), while one-third is extracellular, primarily in the form of interstitial fluid, the fluid surrounding cells.

Only 5% of the total body water is found in the intravascular compartment, meaning it is present in the blood plasma.

Edema is a term used to describe an abnormal accumulation of fluid in the interstitial tissue spaces. When fluid collects in different body cavities, specific terms are used:

\*\*Hydrothorax:\*\* Refers to fluid accumulation in the pleural cavity (around the lungs).
 \*\*Hydropericardium:\*\* Describes fluid accumulation in the pericardial cavity (around the heart).

3. \*\*Hydroperitoneum (Ascites):\*\* Denotes fluid accumulation in the peritoneal cavity (abdominal cavity).

Furthermore, the term \*\*Anasarca\*\* is used to describe severe generalized edema characterized by profound subcutaneous swelling throughout the body. It signifies a widespread and significant fluid retention that goes beyond localized areas or cavities.

## EDEMA

Fluid movement between the vascular & interstitial spaces is governed by two opposing forces, the vascular hydrostatic pressure and the colloid osmotic pressure produced by plasma هدول العوّية هم الي بحدوا استقال الا، إلى الحارج · Normally the outflow of fluid produced by hydrostatic pressure at the arteriolar end of the microcirculation is balanced by the inflow due to the slightly elevated osmotic pressure at the venular end, hence, there is only a small net outflow of fluid into the interstitial spaces, which is drained by the lymphatic vessels. **Either increased hydrostatic pressure or decreased osmotic** pressure causes increased movement of water into the interstitium. - inbalance - Edema. Excess edema fluid is removed by the lymphatic drainage & returned to the bloodstream by the way of the thoracic duct.

Fluid movement between blood vessels (vascular) and the surrounding tissues (interstitial spaces) is influenced by two opposing forces: vascular hydrostatic pressure and colloid osmotic pressure generated by plasma proteins.

Under normal conditions, there is a dynamic balance. The outflow of fluid, driven by hydrostatic pressure at the arteriolar end of the microcirculation, is offset by the inflow due to slightly elevated osmotic pressure at the venular end. As a result, there is typically only a small net outflow of fluid into the interstitial spaces. This fluid is then drained by the lymphatic vessels, preventing excessive accumulation.

However, disruptions to this balance can occur. Increased hydrostatic pressure or decreased osmotic pressure can upset the equilibrium, leading to an increased movement of water into the interstitium. This imbalance contributes to the formation of edema, an abnormal accumulation of fluid in the tissues.

Excess edema fluid is managed by the lymphatic drainage system. Lymphatic vessels collect the accumulated fluid from the interstitial spaces and transport it back to the bloodstream via the thoracic duct. This process helps remove the excess fluid, maintaining the overall fluid balance in the body and preventing the persistence of edema.

On the mechanisms causing edema:

- increased capillary hydrostatic pressure
- decreased plasma oncotic pressure,
  - enhanced permeability of capillary walls(inflammation)
  - 4) Iymphatic obstruction.
    - Each of the types can be further divided into generalized and local forms.

## **FLUID TRANSIT**



Note: (Wikipedia)- Oncotic pressure, or colloid osmotic pressure, is a form of osmotic pressure exerted by proteins, notably albumin, in a blood vessel's plasma (blood/liquid) that usually tends to pull water into the circulatory system. It is the opposing force to capillary filtration pressure and interstitial colloidal osmotic pressure.



The movement of water and low molecular weight solutes such as salts between the intravascular and interstitial spaces is controlled primarily by the opposing effect of vascular hydrostatic pressure and plasma colloid osmotic pressure.

http://faculty.pasadena.edu/dkwon/chapter%2015/chapter%2015\_files/images/image10.png

If the movement of water into tissues (or body cavities) exceeds lymphatic drainage, fluid accumulates.
 An abnormal increase in

interstitial fluid

called edema.

within tissues is

Tonsils Lymph vessels Thymus Spleen Lymph nodes Bone marrow Diagram of the lymphatic system Copyright © CancerHelp UK

## Appearance of edema

- Swollen tissues (not cells—fluid is outside the cells)
- Heavy tissues
- Wet tissues
- Widening of fascial planes or interlobular septa
- Filled cavities